

In the Specification:

Please amend the following paragraphs of the specification to correct misspellings of *Saccharomyces cerevisiae*.

Please amend paragraph [0011] of the published application as follows:

[0011] According to still other embodiments of the present invention, the carboxylase (and corresponding peptide) are from *Magnaporthe grisea*, ~~*Saeearomyces*~~ *Saccharomyces* *cerevisiae* and *Homo sapiens*.

Please amend paragraph [0040] of the published application as follows:

[0040] Described herein is the use of recombinant, isolated, biotin carboxylase domains for the discovery of new Acetyl CoA carboxylase (ACCase) inhibitors. A biotin carboxylase (BC) domain from the ACCase gene of the basidiomycete *Ustilago maydis* was isolated, cloned, expressed, and characterized. The isolated BC domain was shown to have similar high-affinity, soraphen-binding properties as the full-length protein. In contrast to the full-length protein (FIG. 1), however, the BC domain is significantly smaller and can be expressed at higher levels, is more stable, and exists as a monomer. The isolated BC domain is useful for screening new ACCase inhibitors. The BC domain from the oomycete *Phytophthora infestans* was also cloned. A full-length ACCase sequence from this organism has not been published. The appropriate fragment was cloned utilizing PCR using primers derived from published EST's that showed homology to sequences flanking the soraphen-binding domain that was identified in the *Ustilago* gene. The recombinantly expressed *Phytophthora* BC domain exhibited high-affinity soraphen-binding. BC domains from *M. grisea*, *S. cerevisiae cerevisiae*, and *H. sapiens* were also similarly cloned and determined to exhibit high-affinity soraphen-binding, thus demonstrating the applicability of this approach to distantly related organisms.

Please amend paragraph [0079] of the published application as follows:

[0079] In some assays it may be desirable to use a first peptide of the present invention in conjunction (*e.g.*, sequentially or simultaneously) with a second peptide that serves as a counterselection agent. For one embodiment, the counterselection agent may be a peptide of the same species as the first peptide (that is, with substantially the same amino acid sequence as the first peptide), but with a nonfunctional BC domain (for example, by introduction of a deletion or substitution mutation therein), to select against agents that bind non-specifically (*e.g.*, not at the soraphen binding site) to the first peptide. An example would be an *S. cerevisiae* ~~*eerivasae*~~ first peptide and a corresponding *S. cerevisiae* ~~*eerivasae*~~ second peptide in which the second peptide contains a mutation that disrupts soraphen binding (*e.g.*, S77->Y). In another embodiment, the second peptide counterselection agent may be a peptide of a different species as the first peptide, but with a functional BC domain, to detect agents that bind to and act on the first species but not the second species. For example, the first peptide may be non-mammalian, and the second peptide may be mammalian or human (*e.g.*, to select against agents that are active on the mammalian or human ACCase). Where a species contains two different ACCases such as does human, the first and second peptide may be of the same species but a different ACCase (*e.g.*, human ACC1 and human ACC2). In either embodiment, the first and second peptides can be provided together as kits or sets, either *per se* or as compositions/formulations as described above, which may be stored, utilized and/or packaged together, optionally including instructions for their use in assays as described herein.

Please amend paragraph [0095] of the published application as follows:

[0095] Partial summary of BC domains generated. A number of biotin carboxylase (BC) domains that have been characterized herein and can be purified in sufficient quantities for use in affinity based screens or selections, including but not limited to selections using Evolutionary Chemistry, and five BC domains are as follows: wild type versions from *Ustilago maydis*, *Phytophthora infestans*, *Magnaporthe grisea*, and *Saccharomyces cerevisiae*; and the mutated version from *Saccharomyces cerevisiae*. Amino acid sequence alignments of the expressed domains are shown in Figure 10. Identical residues are indicated with an asterisk, and the S to Y mutation in *S. cerevisiae* ~~*eeravisae*~~ that abolishes soraphen binding is indicated in bold.